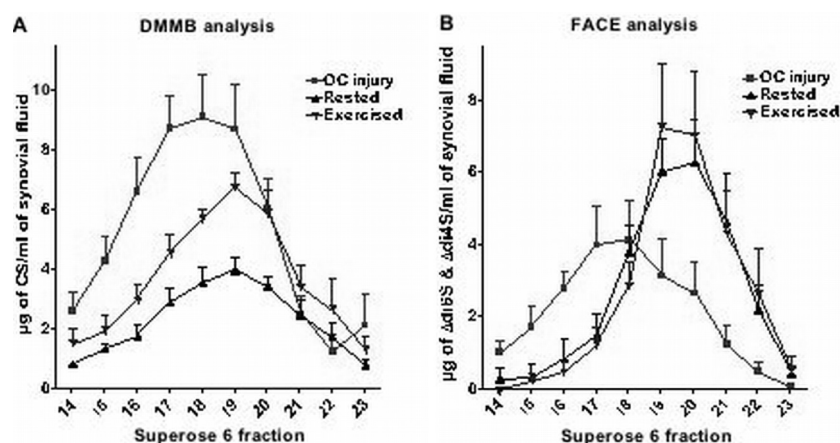


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shows synovial fluid CS content of the 3 groups as determined by DMMB. For comparison, Figure 1B shows total sulfated chondroitin disaccharide (delta di6S and delta di4S) for the same samples determined by FACE. For both methods, CS peak occurs at fraction 18 in synovial fluid from joints with OC injury. Similarly, CS in synovial fluid from exercised joints peaks in fraction 19 with both assays. CS in synovial fluid from rested joints peaks in fraction 19 when determined by DMMB, but peaks in fraction 20 when measured by FACE. When $\mu\text{g/ml}$ amounts of CS for each data point were compared between assays, results for exercised ($n=80$) and OC injury joints ($n=70$) were significantly different ($P<0.0001$), but results for rested joints ($n=80$) were not ($P=0.92$).

Conclusions: When used for quantitation of sulfated CS in synovial fluid ($\mu\text{g/ml}$), DMMB gave results that were significantly different than those from FACE. DMMB may be measuring other sGAGs in addition to CS. However, when performed after Superose 6 chromatography, DMMB assay appears to discriminate the longer CS chains in synovial fluid from OC injured joints from the shorter chains seen in normal joints. Thus, for the specific purposes of determination of CS chain length, the more simple, less expensive DMMB assay may be a replacement for FACE assay. Use of a 384-well microplate allowed use of low volumes of DMMB reagents. This facilitated quantitation of small amounts of CS in fractions collected from Superose 6 chromatography.

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NOVEL CYTOKINE-LIKE MOLECULES RESISTIN AND ADIPONECTIN IN SERUM AND SYNOVIAL FLUID FROM PATIENTS WITH OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS

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Purpose: Adipocytokines resistin and adiponectin are the major members of adipose tissue-derived peptides, which have been suggested as a link between insulin resistance, diabetes, and obesity. Recent data provided also body of evidence on the implication of adipocytokines in the inflammation, immune response, and tissue destruction. This study was undertaken to examine the (patho)physiological role of resistin and adiponectin in matched serum and synovial fluid samples from patients with rheumatoid arthritis (RA) and osteoarthritis (OA).

Methods: Blood and synovial fluid samples from 20 RA and 20 OA patients were analyzed for resistin and adiponectin (ELISA, Biovendor). The clinical activity of patients with OA and RA

was assessed according to the Western Ontario and McMaster Universities (WOMAC) index and the 28 joint count Disease Activity Score (DAS28), respectively.

Results: Serum resistin levels were significantly lower in OA compared to RA patients ($p=0.002$), however the levels of adiponectin in those patients were comparable ($p=0.38$).

The levels of both adipocytokines in synovial fluid were significantly lower in OA than in RA patients ($p<0.001$). Adiponectin concentration was lower in synovial fluid than that in systemic circulation in both diseases. However, in contrast to OA patients, resistin levels were significantly higher in synovial fluid than in systemic circulation in RA patients. In RA patients, increased serum resistin correlated positively with CRP ($r=0.53$, $p<0.02$) and DAS28 ($r=0.44$, $p<0.05$), and adiponectin correlated negatively with leukocyte count in synovial fluid ($r=-0.45$, $p<0.05$). No such correlation was observed in OA patients.

Conclusions: The local up-regulation of adipocytokines in RA synovial fluid points to a possible role of these molecules in the pathophysiology of inflammatory joint diseases such as RA rather than OA.

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INTERLEUKIN-1 ALPHA OR ONCOSTATIN M AND TUMOUR NECROSIS FACTOR ALPHA IN COMBINATION STIMULATE AGGREGANASE BUT NOT MATRIX METALLO PROTEINASE MEDIATED AGGREGAN TURNOVER IN HUMAN ARTICULAR CARTILAGE

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Purpose: Aggrecan is the major proteoglycan in articular cartilage, and is degraded and released from the extracellular matrix in pathological joint debilitating diseases like osteoarthritis (OA). The aggrecanases, ADAMTS-4 and ADAMTS-5, and an array of Matrix Metallo Proteinases (MMPs) are up-regulated in osteoarthritis (OA). The aim of the current study was to monitor the aggrecanase and MMP mediated turnover of aggrecan in human OA cartilage in response to different cytokines.

Methods: Articular cartilage was obtained from OA patients, who had undergone total knee arthroplasty. Articular cartilage was cultured for 21 days with refreshment of medium every 3rd day in the presence of 100 ng/mL Interleukin-1 alpha (IL-1 α) or 10 ng/mL Oncostatin M (OSM) + 20 ng/mL Tumour Necrosis Factor alpha (OSM/TNF α) to stimulate cartilage degradation. As negative control, explants without cytokine stimulation were